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L6	L4 same GAL same yeast	0	L6
L5	L4 same GAL4	0	L5
L4	L1 same (glucocorticoid or estrogen) same receptor	31	L4
L3	L2 same (glucocorticoid or estrogen) same receptor	0	L3
L2	L1 same (ampicillin near0 resistant)	48	L2
L1	cDNA near0 library	12856	L1

L8 ANSWER 1 OF 2 MEDLINE DUPLICATE 1
AN 97265407 MEDLINE
DN 97265407 PubMed ID: 9111344
TI GRIP1, a transcriptional coactivator for the AF-2 transactivation domain of steroid, thyroid, retinoid, and vitamin D receptors.
AU Hong H; Kohli K; Garabedian M J; Stallcup M R
CS Department of Pathology, University of Southern California, Los Angeles 90033, USA.
NC DK43093 (NIDDK)
SO MOLECULAR AND CELLULAR BIOLOGY, (1997 May) 17 (5) 2735-44.
Journal code: 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U39060
EM 199705
ED Entered STN: 19970523
Last Updated on STN: 19970523
Entered Medline: 19970515
AB After binding to enhancer elements, transcription factors require transcriptional coactivator proteins to mediate their stimulation of transcription initiation. A search for possible coactivators for steroid hormone receptors resulted in identification of **glucocorticoid receptor** interacting protein 1 (GRIP1). The complete coding sequence for GRIP1, isolated from a mouse brain **cDNA library**, contains an open reading frame of 1,462 codons. GRIP1 is the probable ortholog of the subsequently identified human protein transcription intermediary factor 2 (TIF2) and is also partially homologous to steroid **receptor** coactivator 1 (SRC-1). The full-length GRIP1 interacted with the hormone binding domains (HBDs) of all five steroid receptors in a hormone-dependent manner and also with HBDs of class II nuclear receptors, including thyroid **receptor** alpha, vitamin D **receptor**, retinoic acid **receptor** alpha, and retinoid X **receptor** alpha. In contrast to agonists, **glucocorticoid** antagonists did not promote interaction between the **glucocorticoid receptor** and GRIP1. In yeast cells, GRIP1 dramatically enhanced the transcriptional activation function of proteins containing the HBDs of any of the above-named receptors fused to the **GAL4** DNA binding domain and thus served as a transcriptional coactivator for them. This finding contrasts with previous reports of TIF2 and SRC-1, which in mammalian cells enhanced the transactivation activities of only a subset of the steroid and nuclear receptors that they physically interacted with. GRIP1 also enhanced the hormone-dependent transactivation activity of intact **glucocorticoid receptor**, **estrogen receptor**, and mineralocorticoid **receptor**. Experiments with **glucocorticoid receptor** truncation and point mutants indicated that GRIP1 interacted with and enhanced the activity of the C-terminal AF-2 but not the N-terminal AF-1 transactivation domain of the **glucocorticoid receptor**. These results demonstrate directly that AF-1 and AF-2 domains accomplish their transactivation activities through different mechanisms: AF-2 requires GRIP1 as a coactivator, but AF-1 does not.

L8 ANSWER 2 OF 2 MEDLINE DUPLICATE 2
AN 96209838 MEDLINE
DN 96209838 PubMed ID: 8643509
TI GRIP1, a novel mouse protein that serves as a transcriptional coactivator in yeast for the hormone binding domains of steroid receptors.
AU Hong H; Kohli K; Trivedi A; Johnson D L; Stallcup M R
CS Department of Pathology, University of Southern California, Los Angeles, 90033, USA.

NC DK43093 (NIDDK)
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 May 14) 93 (10) 4948-52.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U39060
EM 199607
ED Entered STN: 19960726
Last Updated on STN: 19960726
Entered Medline: 19960718

AB The yeast two-hybrid system was used to isolate a clone from a 17-day-old mouse embryo **cDNA library** that codes for a novel 812-aa long protein fragment, **glucocorticoid receptor** -interacting protein 1 (GRIP1), that can interact with the hormone binding domain (HBD) of the **glucocorticoid receptor**. In the yeast two-hybrid system and in vitro, GRIP1 interacted with the HBDs of the **glucocorticoid**, **estrogen**, and androgen receptors in a hormone-regulated manner. When fused to the DNA binding domain of a heterologous protein, the GRIP1 fragment activated a reporter gene containing a suitable enhancer site in yeast cells and in mammalian cells, indicating that GRIP1 contains a transcriptional activation domain. Overexpression of the GRIP1 fragment in mammalian cells interfered with hormone-regulated expression of mouse mammary tumor virus-chloramphenicol acetyltransferase gene and constitutive expression of cytomegalovirus-beta-galactosidase reporter gene, but not constitutive expression from a tRNA gene promoter. This selective squelching activity suggests that GRIM can interact with an essential component of the RNA polymerase II transcription machinery. Finally, while a steroid **receptor** HBD fused with a **GAL4** DNA binding domain did not, by itself, activate transcription of a reporter gene in yeast, coexpression of this fusion protein with GRIP1 strongly activated the reporter gene. Thus, in yeast, GRIP1 can serve as a coactivator, potentiating the transactivation functions in steroid **receptor** HBDs, possibly by acting as a bridge between HBDs of the receptors and the basal transcription machinery.

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FILE 'GENBANK' ENTERED AT 13:51:15 ON 27 JAN 2003

L1 5 S AF124093

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 13:57:10 ON 27 JAN 2003

L2 54023 S CDNA (W)LIBRARY
L3 9 S L2 (P)(AMPICILLIN (W)RESISTANT)
L4 285 S L2 (P)(GLUCOCORTICOID OR ESTROGEN) (P) RECEPTOR
L5 0 S L4 (P)(AMPICILLIN OR KANAMYCIN)
L6 0 S L4 (P)GAL (P)YEAST
L7 8 S L4 (P)GAL4
L8 2 DUPLICATE REMOVE L7 (6 DUPLICATES REMOVED)